AD-A036 038

CARDIAC ARRHYTHMIAS IN EXPERIMENTAL SYNCOPE

SCHOOL OF AVIATION MEDICINE
RANDOLPH AIR FORCE BASE, TEXAS

NOVEMBER 1958

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Fifty young, apparently healthy aviation cadets in preflight training were evaluated with regard to syncope. Thirty percent of these subjects admitted that they had experienced previous, undisclosed syncope, when they were not in fear of detection or removal from the training program. Twenty-one of the cadets experienced experimental syncope during the syncope procedures. Cardiac arrhythmia was frequently associated with the syncopal episode and cardiac arrhythmia was frequently induced by respiratory maneuvers without syncope. Intravenous administration of atropine apparently prevented recurrence of cardiac arrhythmia induced by respiratory maneuvers. Significant cardiac arrhythmia was also noted in simple orthostatic syncope.

Loss of consciousness presents a serious problem in modern aviation and in critical areas of industry. Syncope is commonly seen at the blood bank, during inoculations, in the dentist's chair, and during moments of stress. Although it often occurs, little information is available concerning the likelihood of its recurrence in any individual or its actual relationship to experimental syncope. It is common practice to study the problem of syncope with orthostatic measures utilizing the tilt-table or eliciting well-known reflexes such as the reflex resulting from carotid sinus massage.

Interest in this problem began with the discovery of significant cardiac arrhythmias occurring with simple respiratory maneuvers (1, 2). Some of these were associated with syncope. The respiratory system must make constant adaptations to changes in the environment associated with flight and altitude exposure. This facet of information as related to syncope seemed worthy of further exploration. Eighty-two apparently healthy persons who were engaged in flying activity and who had experienced syncope were studied, 55 of these with special procedures (3). It was impressive to note the number of cardiac arrhythmias that occurred in this series and it was equally impressive to note the relation of cardiac arrhythmias to the experimental production of syncope. Of the group studied by experimental procedures, breathing maneuvers such as breath-holding at the height of inspiration and breath-holding following hyperventilation were most rewarding in illustrating the relation of cardiac arrhythmia to syncope. In the previous study, all the subjects evaluated had admitted to episodes of syncope, thereby placing their future flying careers in jeopardy. It was felt that it would be informative to study, by a similar battery of tests, a group of young flying personnel who had previously denied having experienced syncope. This would also provide an opportunity to more carefully evaluate the role of atropine both in creating and in preventing syncope in such a group. Atropine was used in only a few instances in the previous study.

Received for publication on 4 August 1958.

METHODS AND MATERIALS

Fifty healthy cadets in preflight training were brought to the School of Aviation Medicine for syncope evaluation. They were volunteers who were told only that they would serve as experimental subjects for tests at the School of Aviation Medicine. The nature and purpose of tests were not disclosed in advance. As an incentive, each subject was guaranteed extra time off from duty. The cadets represented a relatively young age group. Their ages ranged from 19 years to 27 years, the average being 21.1 years.

All of these individuals had been previously examined and all had denied previous syncopal episodes in order to gain admittance to the

Aviation Cadet Flying Training Program. They were subjected to a battery of special procedures to test the cardiovascular system. This included observation of the patient's blood pressure, pulse, symptoms, and signs at rest and on a tilt-table. The subjects performed breathing maneuvers which included: (1) breath-holding at the height of maximum inspiration without bearing down; (2) prolonged breath-holding; (3) hyperventilation; and (4) breath-holding following hyperventilation. These maneuvers were repeated with the subject in the standing and in the recumbent positions. The carotid sinuses were massaged for 15 seconds and the subject underwent a 15-minute orthostatic tolerance test on the tilt-table. Following these procedures, the subject received atropine, grain $\frac{1}{100}$, and the respiratory maneuvers were repeated.

Each subject was identified by a number which he had drawn from a hat. He was assured that no attempt would be made to identify the number with his name. He was then asked to report whether he had truly had any syncopal episodes in the past and what the circumstances surrounding these were. The purpose of this was to detect, if possible, the number of individuals entering a flying training program who intentionally suppressed information relative to previous syncopal episodes in order to be accepted.

RESULTS

One of the most interesting facets of this study was learning the true incidence of syncope in such a group. Of the 50 aviation cadets, 15 admitted to previous undisclosed syncope.

During the experimental procedures, 21 of the subjects had one or more syncopal episodes, or near enough to syncope to stop the precipitating cause. Of the group having experimental syncope, 15 had significant cardiac arrhythmias associated with the syncopal episode. Two of the subjects having cardiac arrhythmias associated with syncope also had a significant drop in blood pressure at the onset of the arrhythmia. In 7 subjects, breath-holding produced syncope with arrhythmia. One subject had syncope with arrhythmia secondary to breath-holding following hyperventilation. In 5 subjects, carotid sinus stimulation produced arrhythmia associated with syncope. Five of the 15 subjects had syncope associated with arrhythmia due to orthostatic influences. This group represented all cases of syncope due to orthostatic influences. The apparent discrepancy in numbers is due to the fact that a few subjects had syncope from more than one precipitating factor.

Syncope may occur with breathing maneuvers in the absence of apparent significant cardiac arrhythmia. There were 11 subjects in all who had syncope with breath-holding maneuvers and an additional subject who had syncope with breath-holding following hyperventilation. Often the syncopal episode occurred during the recovery phase after breath-holding. The true incidence of syncopal episodes which might be precipitated by such breathing maneuvers is slightly higher than this, as those individuals who had strong orthostatic influences were sometimes exempted from the remainder of the experimental protocol. This procedure was followed because these individuals could not be properly evaluated concerning their response to specific stimuli. In 9 of the 21 subjects, syncope could be induced with carotid sinus massage (5 associated with cardiac arrhythmia and 4 without arrhythmia). Syncope commonly occurred after, rather than during, massage. In 5 of the subjects syncope could be induced by orthostatic influences (all 5 were associated with arrhythmia).

Of the 15 cadets who admitted having had syncopal episodes which they had not previously disclosed, 7 developed experimental syncope during the procedure. This, of course, means that 8 other subjects who had previous clinical syncope had no syncopal episodes during the experimental procedures. Fourteen subjects who experienced syncope under experimental conditions denied any previous episode of clinical syncope.

None of the incidents of syncope in this series were induced by having the subjects holding the breath at the height of inspiration. All syncopal episodes secondary to breathing maneuvers were due to prolonged breath-holding and/or breath-holding following a period of hyperventilation. Breath-holding following hyperventilation commonly produced effects similar to those caused by prolonged breath-holding. Therefore, it is listed in a separate category

only when it alone, as an additional stress mechanism, induced a syncopal episode.

Of the 50 subjects undergoing the experimental syncope procedures, 37 demonstrated some form of cardiac arrhythmia. In 22 instances, the cardiac arrhythmia occurred without subsequent syncope. More than one form of stress was capable of producing arrhythmia in a number of the individuals. In 26 subjects, cardiac arrhythmia of some type could be induced by breath-holding and by breath-holding following hyperventilation. In 2 additional cases, cardiac arrhythmia was induced only by breath-holding following hyperventilation. Twenty-eight of the cadets had some form of cardiac arrhythmia following one of the respiratory maneuvers. Eight cadets demonstrated cardiac arrhythmias following carotid sinus massage. Five cadets developed cardiac arrhythmia associated with orthostatic stress. It is interesting to note that of the 7 subjects who had a previous clinical episode of syncope as well as having syncope during the experimental routine, 6 had syncope secondary to breath-holding and 1 subject experienced it because of orthostatic influences.

In 30 instances in which a cardiac arrhythmia was induced by respiratory maneuvers, atropine was administered. Following adequate atropinization (approximately 6 minutes after intravenous injection of grain 1/50 of atropine), cardiac arrhythmias were not induced by any of the respiratory maneuvers.

A very brief summary of the findings in the individual cases is presented in table I.

DISCUSSION

The incidence of syncope in this group of cadets beginning flying training suggests that the reported low incidence of syncope in cadets is entirely fictitious and represents a suppression of information by the cadet applicants in order to be admitted to the program. If a similarly high incidence of syncope should exist in all of the individuals accepted for preflight training, it may be assumed that between 18 and 42 percent of all pilots beginning flying training have already experienced some form of clinical syncope.[1] Needless to say, a large percentage of these individuals complete their flying training and carry out to completion a useful flying career.

The relatively high incidence of syncope in a normal aviation cadet preflight group is a strong argument against the concept that syncope itself presents an underlying disease state. It is difficult to assume that 30 percent of our highly selected, apparently healthy young men have an underlying disease process or disease complex which might prove hazardous to a career in flying or in crucial industry.

In this series, it was impressive to note the high incidence of cardiac arrhythmias associated with syncopal episodes. Cardiac arrhythmias appeared to be important even in the presence of experimental orthostatic syncope. While it had been previously noted that cardiac arrhythmias were important in syncope induced by respiratory maneuvers (2), this finding in orthostatic syncope suggests their importance in other forms of syncope as well (figs. 1 and 2). Subjects 5, 17, 31, 35, and 47 demonstrated significant cardiac arrhythmia in association with their syncopal episodes.

In cases of syncope induced by carotid sinus massage, both cardiac arrhythmias and vasodepressor response were commonly presented (fig. 3).

The respiratory maneuvers presented the largest number and the largest variety of cardiac arrhythmias. Passive rhythms, including atrial rhythm, A-V dissociation with nodal rhythm, and lower nodal rhythm were commonly noted (figs. 4 and 5). One episode of nodal tachycardia was also observed (fig. 6).

In some instances cardiac arrhythmias were distinctly obvious as the major factor precipitating a syncopal episode. In the presence of cardiac arrest with no cardiac beat, no alternative explanation seems feasible. In those cardiac arrhythmias which resulted in marked depression of the cardiac rate, it might be assumed that the accompanying cardiac arrhythmia in the presence of other factors was sufficient to create syncope. In the event that any degree of vasodepressor syncope is present with peripheral arteriolar dilatation, progressive depression of the cardiac rate further compromises cardiac output. This, in turn, can be expected to lead to cerebral ischemia and induce a syncopal episode. As indicated in

[1] The observed proportion of 30 percent is a sample estimate of the true proportion in the total population of pilots beginning flying training. As such, it is subject to sampling variation. Using a confidence interval of 2, it can be expected that there is only 1 chance in 20 that the true proportion having an episode of syncope lies below 18 or above 42 percent.

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TABLE I

Clinical study of 50 aviation cadets

Subject No.	Age (years)	Clinical history	Experimental findings
1	20	None.	Breath-holding caused a shift of the pacemaker to an atrial focus. Subsequent to atropinization,* breath-holding failed to produce a shift in cardiac pacemaker.
2	20	Syncope following bilateral carotid occlusion and syncope 3 hours following immunization.	Breath-holding caused a shift in the pacemaker to an atrial focus. Once precipitated, other respiratory maneuvers could produce a shift in the pacemaker. Two atrial premature contractions were noted. Following atropinization, no arrhythmia was noted.
3	22	None.	Breath-holding resulted in sinus bradycardia. Carotid sinus massage caused marked slowing to 38 beats per minute associated with a near syncopal episode. Two minutes following carotid sinus massage, the rhythm changed to nodal rhythm, then to normal sinus rhythm, then to A-V dissociation. Hyperventilation followed by breath-holding induced atrial rhythm. Following atropinization no changes in pacemaker or cardiac rhythm could be induced.
4	22	None.	No findings.
5	23	At age 13 he fainted after breaking his finger.	During baseline studies, when first placed on a tilt-table the subject experienced 3 seconds of asystole followed by escape, followed by another 6-second period of asystole and escape. After recurrent episodes of asystole, the patient developed nodal rhythm, and syncope occurred.
6	23	None.	Immediately following left carotid sinus massage, the patient felt weak and faint. Two minutes later he developed a period of asystole followed by syncope. Asystole was terminated by ventricular escape beat, a short period of atrial flutter, and, finally, resumption of normal sinus rhythm. Following breath-holding, A-V dissociation with nodal rhythm occurred. Following atropinization no arrhythmia could be elicited by breathing maneuvers.
7	20	None.	No findings.
8	20	None.	Following prolonged breath-holding, there was a change in the PR interval to 0.08 second with upright T-waves in all leads. Following atropinization breath-holding produced no changes from normal sinus rhythm.
9	20	Syncope at age 12 induced by chest squeeze during breath-holding following a period of hyperventilation.	Prolonged breath-holding caused change to an atrial focus. At the height of inspiration with breath-holding, there was a period of second-degree A-V block. Prolonged breath-holding was followed by episodes of second-degree A-V block and syncope. Following atropinization breath-holding produced no changes from normal sinus rhythm.

TABLE I (Contd.)

Subject No.	Age (years)	Clinical history	Experimental findings
10	20	At age 18 he had syncope during a chest squeeze in full inspiration following hyperventilation.	Breath-holding caused a shift to an atrial focus and atrial rhythm. Breath-holding following hyperventilation induced one ventricular premature contraction. Following adequate atropinization, no arrhythmias could be induced by breathing maneuvers.
11	20	None.	Following breath-holding, atrial rhythm appeared. Following atropinization no arrhythmia could be induced.
12	22	None.	Following breath-holding there was a shift in the pacemaker to an atrial focus followed by bradycardia which persisted for 2¼ minutes, then A-V dissociation, nodal rhythm, and syncope. Following atropinization no arrhythmia could be induced by breathing maneuvers.
13	21	Age 16. He had been sitting with his feet up on the stove; he got up, yawned, and passed out.	Breath-holding caused a shift in the pacemaker to a secondary atrial focus.
14	20	None.	Right carotid sinus massage caused a change in the pacemaker to a secondary site. Two minutes following massage, the patient had a near syncopal episode. Breath-holding caused a shift in the pacemaker to a secondary atrial focus. Following atropinization no arrhythmia could be induced by breathing maneuvers.
15	22	At age 20, he had syncope associated with orthostatic hypertension.	On release of breath following prolonged breath-holding, one ventricular premature contraction was noted. During hyperventilation, one ventricular prematurity was noted and after tilt to the horizontal position, one atrial premature contraction occurred. Following adequate atropinization no arrhythmias were noted.
16	21	None.	Right carotid sinus massage caused a change in the pacemaker to a secondary atrial focus. Following breath-holding, atrial rhythm and atrial fusion beats were noted. Breath-holding after hyperventilation produced an atrial rhythm with second-degree A-V block, and A-V dissociation with syncope. Breath-holding with the patient in the recumbent position also produced atrial rhythm. Following atropinization, breath-holding produced no changes from normal sinus rhythm.
17	19	None.	With the patient first placed on the tilt-table to obtain baseline blood pressures and studies, A-V dissociation with nodal rhythm occurred and was associated with syncope.

TABLE I (Contd.)

Subject No.	Age (years)	Clinical history	Experimental findings
18	21	Syncope at age 18. He had influenza and had been marching in the sun.	Breath-holding following hyperventilation caused a drop in blood pressure with progressive slowing of the cardiac rate and syncope.
19	20	Syncope at 8 years of age following an injection.	Following prolonged breath-holding there was A-V dissociation and nodal rhythm (rate 40/min.), followed by syncope. Following atropinization, breath-holding produced no changes from normal sinus rhythm.
20	23	None.	Breath-holding caused a shift in the pacemaker to a secondary atrial focus. Following atropinization, breath-holding produced no changes from normal sinus rhythm.
21	21	None.	Breath-holding produced an immediate slowing of the pacemaker and a change to an atrial rhythm. This was successively repeated with breath-holding. Following atropinization, breath-holding produced no changes from normal sinus rhythm.
22	21	None.	Occasional ventricular premature contractions throughout study.
23	21	None.	Breath-holding resulted in a shift in the pacemaker to a secondary atrial focus. Following atropinization, breath-holding produced no changes from normal sinus rhythm.
24	21	None.	No findings.
25	20	None.	Breath-holding caused a shift in the pacemaker to a secondary site. Following atropinization, breath-holding produced no changes from normal sinus rhythm.
26	20	None.	Left carotid sinus massage was followed by sinus arrest with nodal escape beat. Breath-holding caused sinus bradycardia with A-V block and nodal escape associated with a drop in blood pressure and syncope. Thereafter every breath-holding episode could cause a change in the pacemaker to an atrial focus. Following atropinization, dissociation and changes in the pacemaker were noted. After adequate time for complete atropinization, the patient entered a normal sinus rhythm which persisted despite breathing maneuvers.
27	22	None.	Breath-holding resulted in a shift in the pacemaker to a secondary atrial focus. Following atropinization, breath-holding produced no changes from normal sinus rhythm.
28	21	None.	During breath-holding the patient developed A-V dissociation, felt weak, but recovered. Following atropinization, breath-holding produced no changes from normal sinus rhythm.

TABLE I (Contd.)

Subject No.	Age (years)	Clinical history	Experimental findings
29	21	None.	No findings.
30	26	None.	Breath-holding resulted in a shift in the pacemaker to a secondary site in the atria. Following atropinization, shifts in the pacemaker could not be induced.
31	20	None.	Breath-holding was associated with cardiac slowing and in the recovery period, A-V dissociation and nodal rhythm occurred, the cardiac rate dropping to 44 beats per minute. Syncope resulted. The patient rested in the recumbent position and was then tilted to the vertical position. This resulted in progressive cardiac slowing, a short period of asystole followed by nodal escape and syncope. Atropinization was not followed sufficiently long for evaluation of atropine effect.
32	21	None.	No findings.
33	21	None.	Breath-holding resulted in cardiac slowing, A-V dissociation, nodal rhythm. Breath-holding following hyperventilation resulted in lower nodal rhythm, A-V dissociation. Following atropinization, breath-holding produced no changes from normal sinus rhythm.
34	21	At 16 and 17 years the patient had episodes of syncope following long periods of reading or watching television.	Breath-holding after hyperventilation resulted in a shift in the pacemaker to secondary atrial focus. This was also noted with breath-holding without hyperventilation. Following atropinization, breath-holding produced no changes from normal sinus rhythm.
35	21	None.	At the onset of the baseline studies with the patient first assuming the position on the tilt-table, there was a shift in the pacemaker to nodal rhythm followed by a 5-second period of asystole and syncope After the recovery period, breath-holding would produce A-V dissociation and nodal rhythm. Near syncope occurred during the recovery period following breath-holding. After atropinization, normal sinus rhythm developed and no further arrhythmias or syncope could be induced.
36	22	None.	One minute following right carotid sinus massage there was a sinus bradycardia followed by a shift in the pacemaker to an atrial, then nodal, focus, associated with a near syncopal episode. Following recovery, breath-holding alone was associated with marked cardiac slowing and near syncope. Following atropinization, breath-holding produced no changes from normal sinus rhythm.
37	22	None.	No findings.

TABLE I (Contd.)

Subject No.	Age (years)	Clinical history	Experimental findings
38	23	None.	Following right carotid sinus massage, there was a marked decrease in blood pressure with sinus bradycardia (rate 47/min.) and syncope.
39	21	Syncope occurred while donating blood.	Breath-holding resulted in cardiac slowing with shift in the pacemaker to a secondary atrial site. During prolonged breath-holding, there was a 2-second period of asystole associated with marked decrease in blood pressure and near syncope. Following atropinization, breath-holding produced no changes from normal sinus rhythm.
40	20	Syncope occurred following injection of novocain.	Breath-holding resulted in two atrial premature contractions. Breath-holding following hyperventilation resulted in A-V dissociation, nodal rhythm, and occasional atrial premature contractions. Following atropinization, breath-holding produced no changes from normal sinus rhythm.
41	27	Syncope induced by blowing against his thumb following a period of hyperventilation.	Breath-holding resulted in A-V dissociation with nodal rhythm. Breath-holding following hyperventilation again resulted in A-V dissociation with nodal rhythm. Upon expiration, nodal tachycardia ensued associated with a near syncopal episode. Following atropinization, breath-holding produced no changes from normal sinus rhythm.
42	22	None.	Breath-holding resulted in a shift in the pacemaker to a secondary atrial focus. Following atropinization, breath-holding produced no changes from normal sinus rhythm.
43	20	None.	Breath-holding resulted in A-V dissociation and nodal rhythm. Following atropinization, breath-holding produced no changes from normal sinus rhythm.
44	20	None.	Left carotid sinus massage resulted in cardiac slowing with a shift in the pacemaker to a secondary focus, atrial rhythm, nodal beat, and asystole followed by syncope. Thereafter repeated episodes of breath-holding resulted in shift of the pacemaker to a secondary atrial focus with atrial rhythm, A-V dissociation, and nodal rhythm. Following atropinization, breath-holding produced no changes from normal sinus rhythm.
45	21	None.	Two minutes following right carotid sinus massage, first-degree A-V block occurred followed by second-degree A-V block, with an 8-second period of no ventricular response followed by syncope.
46	20	"Self-initiated" syncope.	On every occasion immediately after release of breath, the patient developed A-V dissociation with block. This did not recur following administration of atropine.

TABLE I (Contd.)

Subject No.	Age (years)	Clinical history	Experimental findings
47	19	Syncope at age 14 while laceration was being sutured and, on another occasion, following venipuncture.	At the onset of baseline studies when the patient first assumed the position on a tilt-table, A-V dissociation with nodal rhythm occurred followed by syncope. Forty-one seconds following right carotid sinus massage, there was a period of asystole followed by syncope. Breath-holding was not done in this instance.
48	21	None.	Breath-holding resulted in a shift of the pacemaker to secondary atrial focus. Thirty seconds after left carotid sinus massage, there was marked cardiac slowing with nodal rhythm and syncope. Thereafter breath-holding produced nodal rhythm with syncope. Following atropinization, breath-holding produced no changes from normal sinus rhythm.
49	21	None.	No findings.
50	19	Syncope at the 1¾ mile mark while running a 2-mile race.	No findings.

*Frequent arrhythmias (e.g., A-V dissociation) were noted immediately after the injection of atropine, due to the initial atropine effect. All maneuvers to test full atropinization were performed at 6 minutes or more after intravenous administration in order to avoid confusing early transitory effects of atropine with the syncope procedures.

a previous paper (3), the respiratory reflexes which originate from stretch receptors within the lungs stimulate the same efferent reflex pathways that are stimulated by the carotid sinus afferent nerves. Thus, one should expect both vasodepressor and cardiac arrhythmia responses when the respiratory stretch reflex is stimulated. In this series, the cardiac arrhythmias seem to be a major factor.

Another interesting finding was that cardiac arrhythmias were not induced in any case by respiratory maneuvers following the administration of atropine. This strongly suggests that adequate atropinization provides a protective mechanism against the development of cardiac arrhythmias by stress procedures. It follows that previous experimentation which has utilized orthostatic tests with the concomitant administration of atropine has protected the experimental subject from demonstrating cardiac arrhythmias (4, 5). It seems that not only may atropine act to produce syncope by creating visceral pooling, but it may also prevent syncope by abolishing vagal reflexes which cause cardiac inhibition and result in cardiac arrhythmia. The intravenous administration of atropine warded off impending syncopal episodes on several occasions in subjects of this series.

One might ask what role shifts in electrolytes might have played in the cardiac arrhythmias precipitated by respiratory maneuvers. It is pertinent to point out that cardiac arrhythmias were induced in subjects by prolonged breath-holding and also by breath-holding at the height of inspiration following hyperventilation. In the former instance with prolonged breath-holding, carbon dioxide retention should result in a tendency toward respiratory acidosis. In the latter instance, hyperventilation should tend toward respiratory alkalosis. Regardless of these two extremes in the assumed change in electrolyte pattern, cardiac arrhythmia was induced.

A point of some interest in this series was the absence of syncope occurring immediately with breath-holding at the height of inspiration. Two subjects in the previously reported series of syncope patients (3) did present this finding.

The subjects of the current study are not entirely comparable to those previously studied

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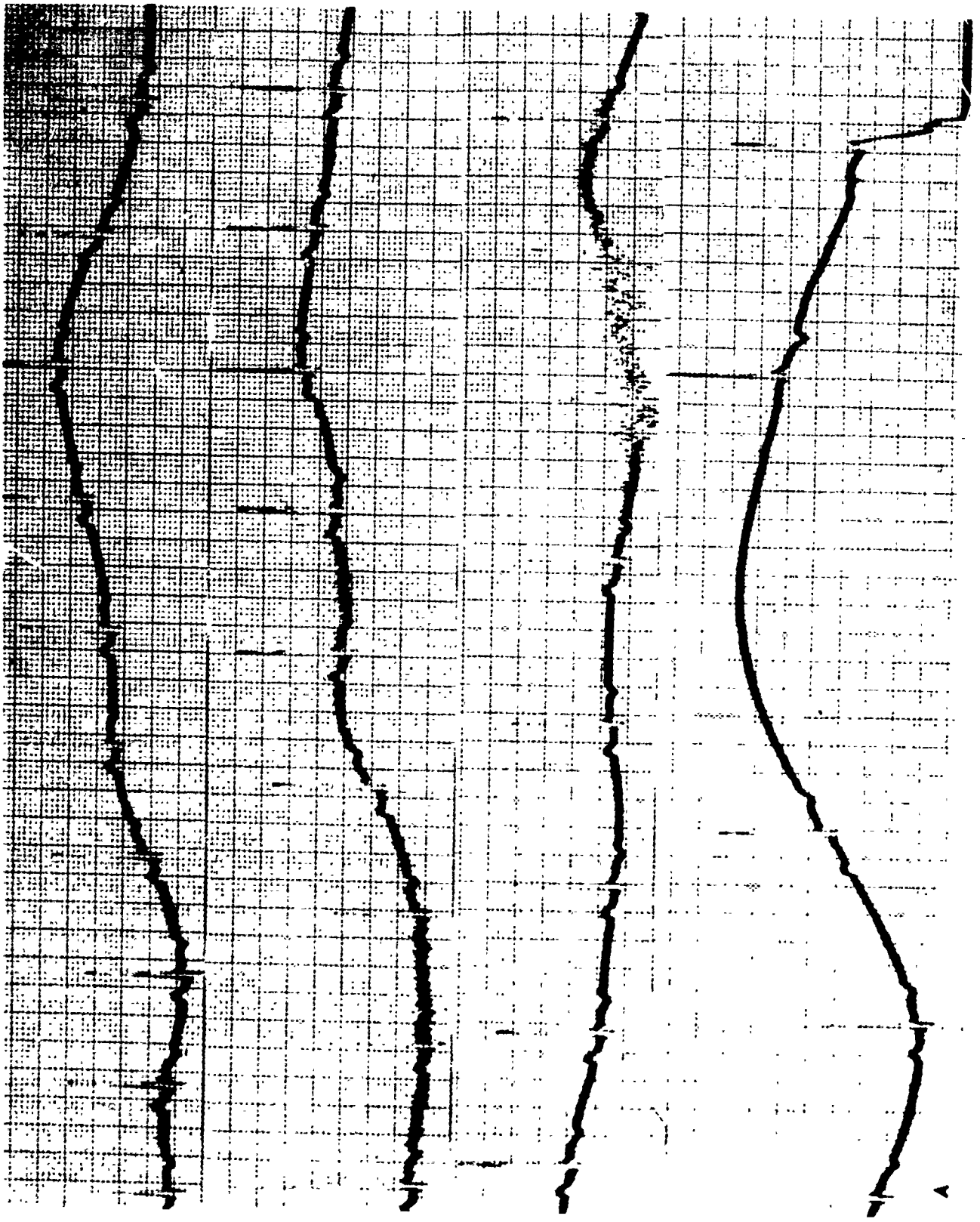


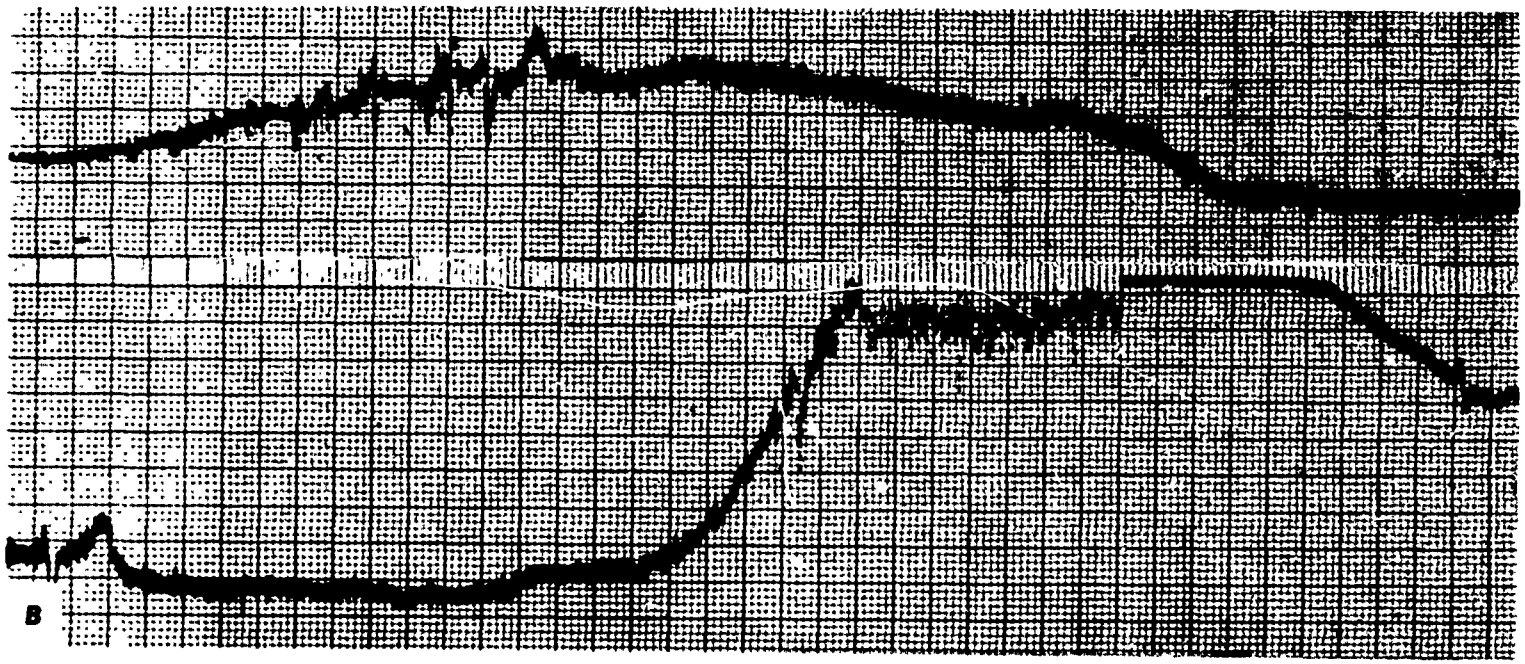


FIGURE 1

A. *Subject 31: A continuous lead II shows progressive slowing, with eventual sinus arrest and nodal escape. This was the result of orthostatic influences.*

B. *Subject 5: This is a continuous lead III taken within 3 minutes after standing on the tilt-table (upright 90°). Sinus arrest with escape beats occurred and syncope followed. Artifact of muscle tremor is present. The subject was tilted to the recumbent position and recovered.*

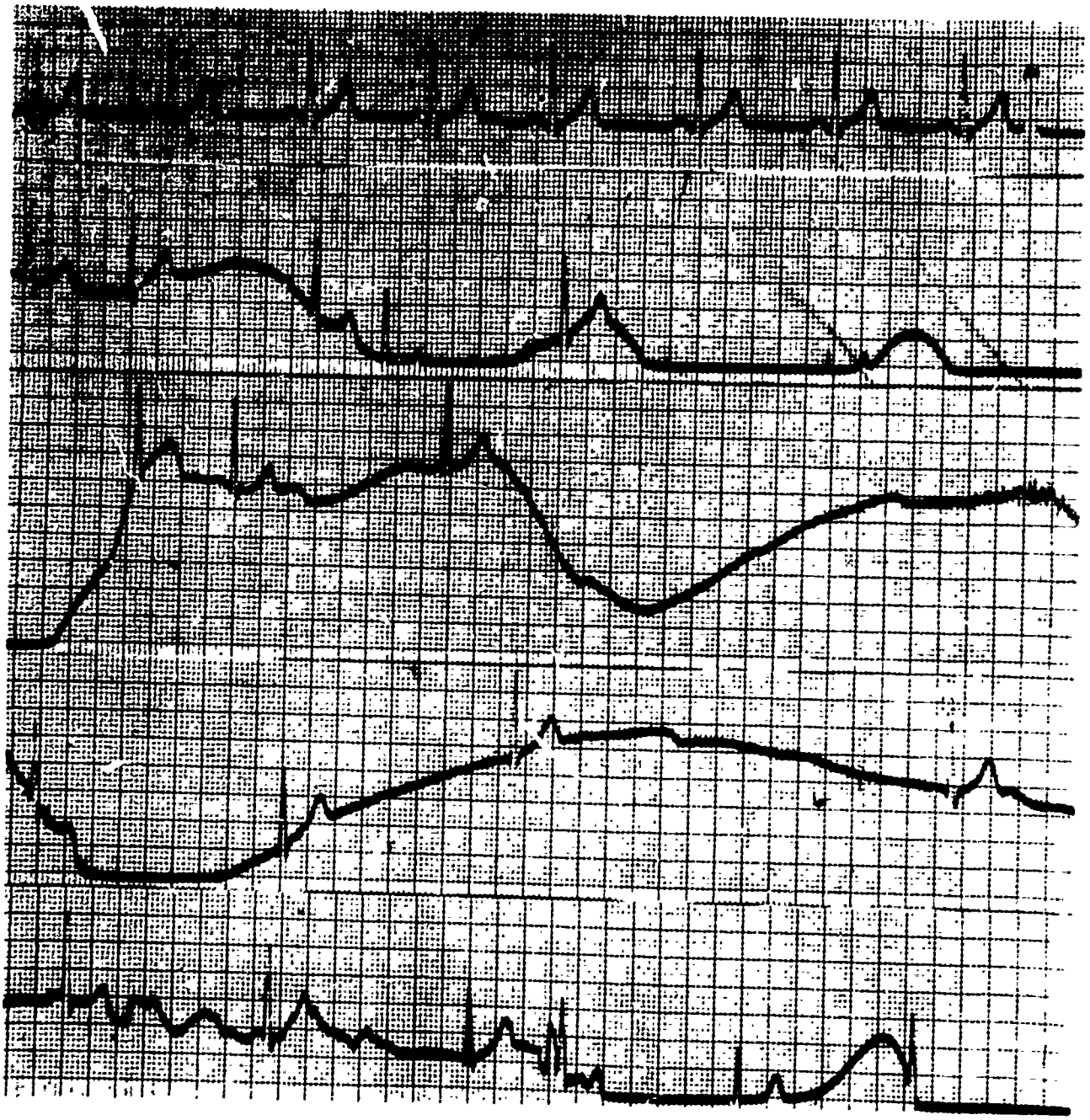


FIGURE 2

Subject 35: The top strip demonstrates baseline lead II in the recumbent position. The remaining tracing is a continuous lead II within 3 minutes after being tilted to upright (90°) position. Sinus arrest with nodal escape and slow nodal rhythm is demonstrated. This occurred at the onset of baseline studies to begin the procedures.

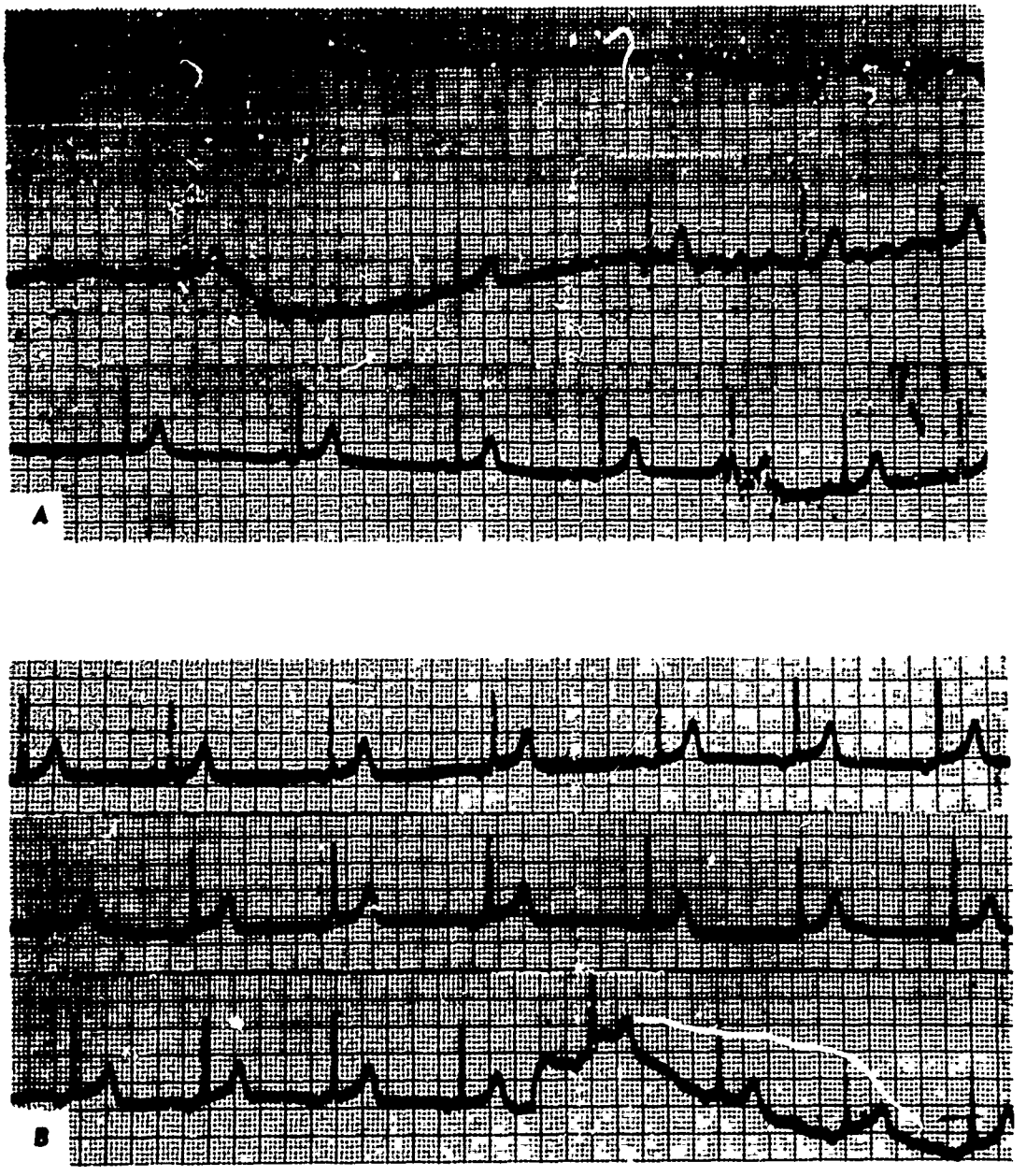


FIGURE 3

A. *Subject 6: Continuous lead II demonstrates period of cardiac arrest terminated by escape beats and transitory atrial flutter. This episode occurred 2 minutes after carotid sinus massage with the patient standing on the tilt-table (90°).*

B. *Subject 6: Continuous lead II after tilting to the horizontal position demonstrates atrial rhythm. The second strip shows atrial rhythm with A-V dissociation. Note inverted P-wave just emerging before the last QRS event.*

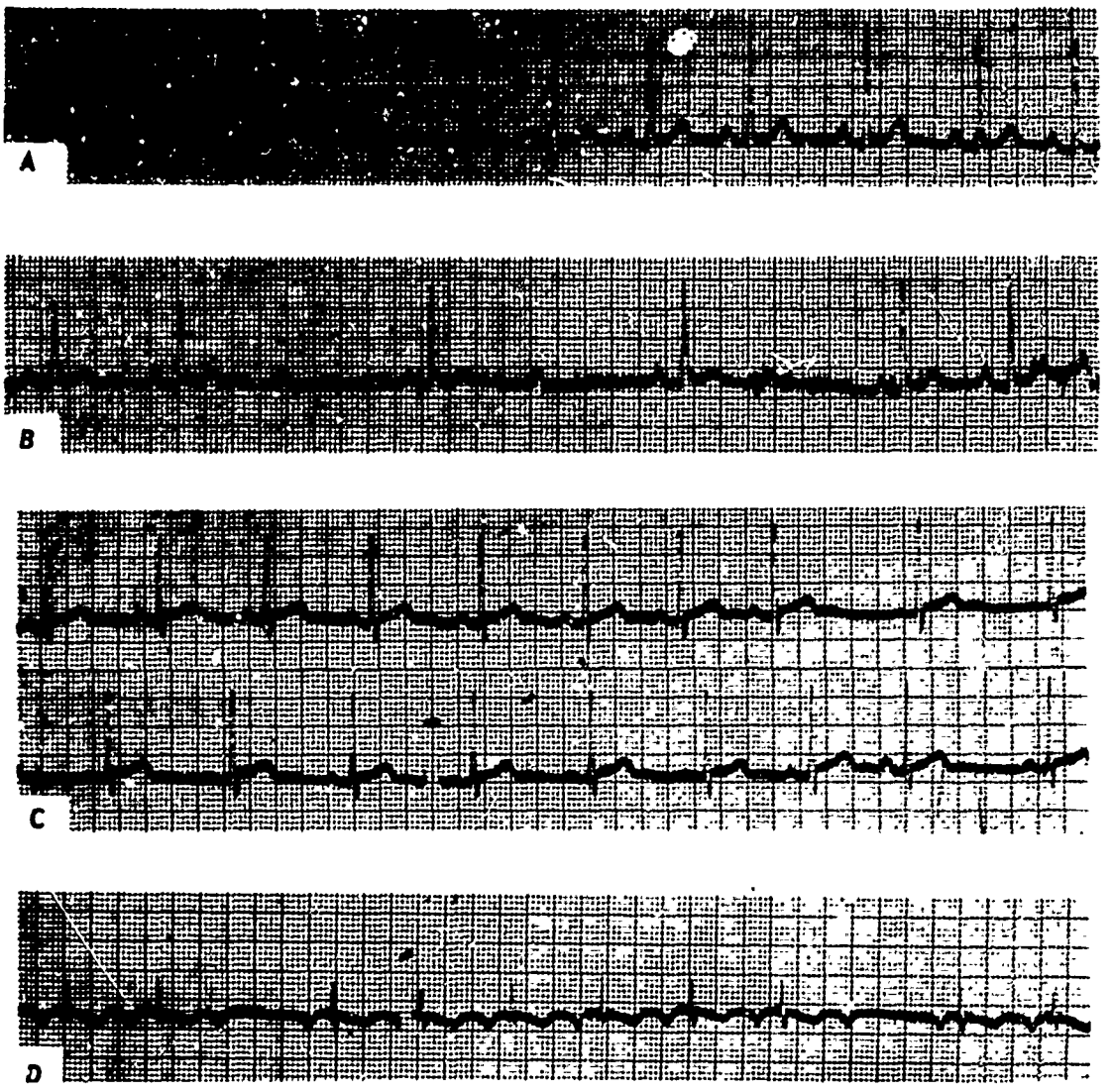


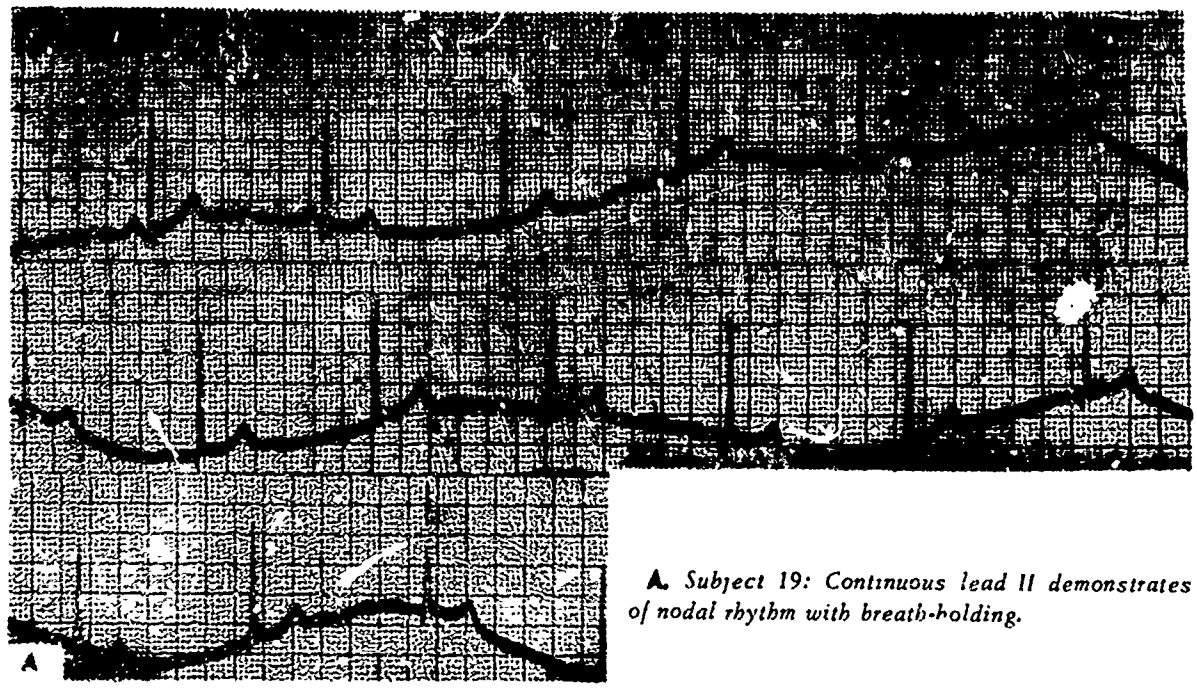
FIGURE 4

A. *Subject 9: A-V block occurs just at the height of inspiration and onset of breath-holding*

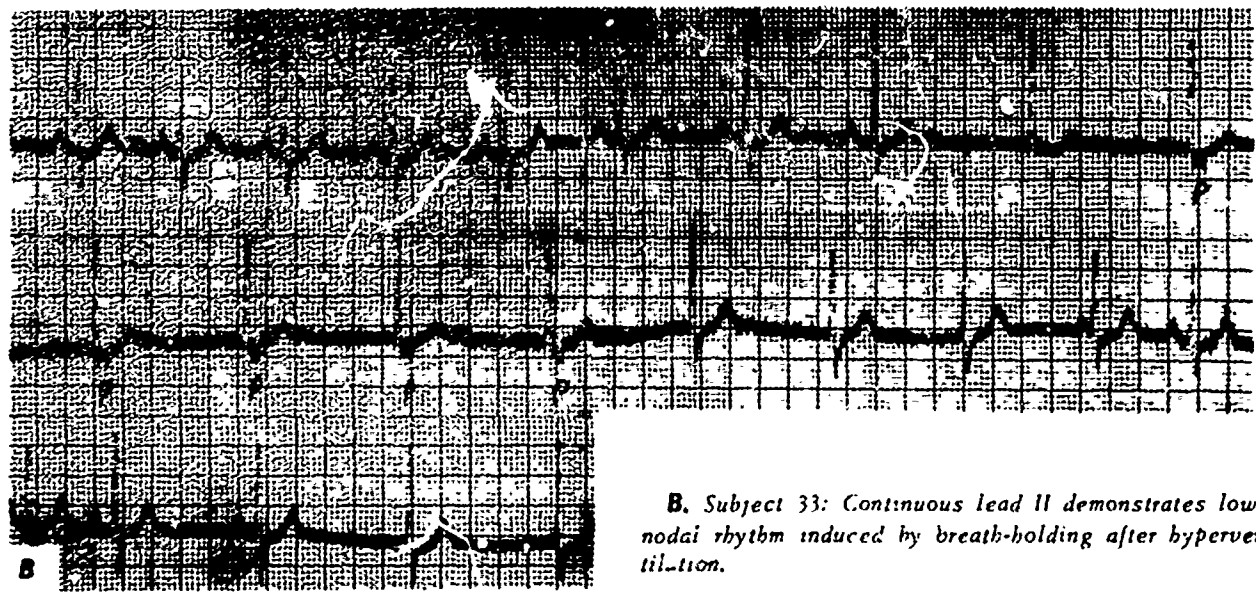
B. *Subject 9: More persistent second degree A-V block during prolonged breath-holding.*

C. *Subject 10: Continuous lead II demonstrates a change in the pacemaker occurring with breath-holding.*

D. *Subject 3: Lead III demonstrates atrial rhythm and A-V block precipitated by breath-holding following hyperventilation.*



A. *Subject 19: Continuous lead II demonstrates onset of nodal rhythm with breath-holding.*



B. *Subject 33: Continuous lead II demonstrates lower nodal rhythm induced by breath-holding after hyperventilation.*

FIGURE 5

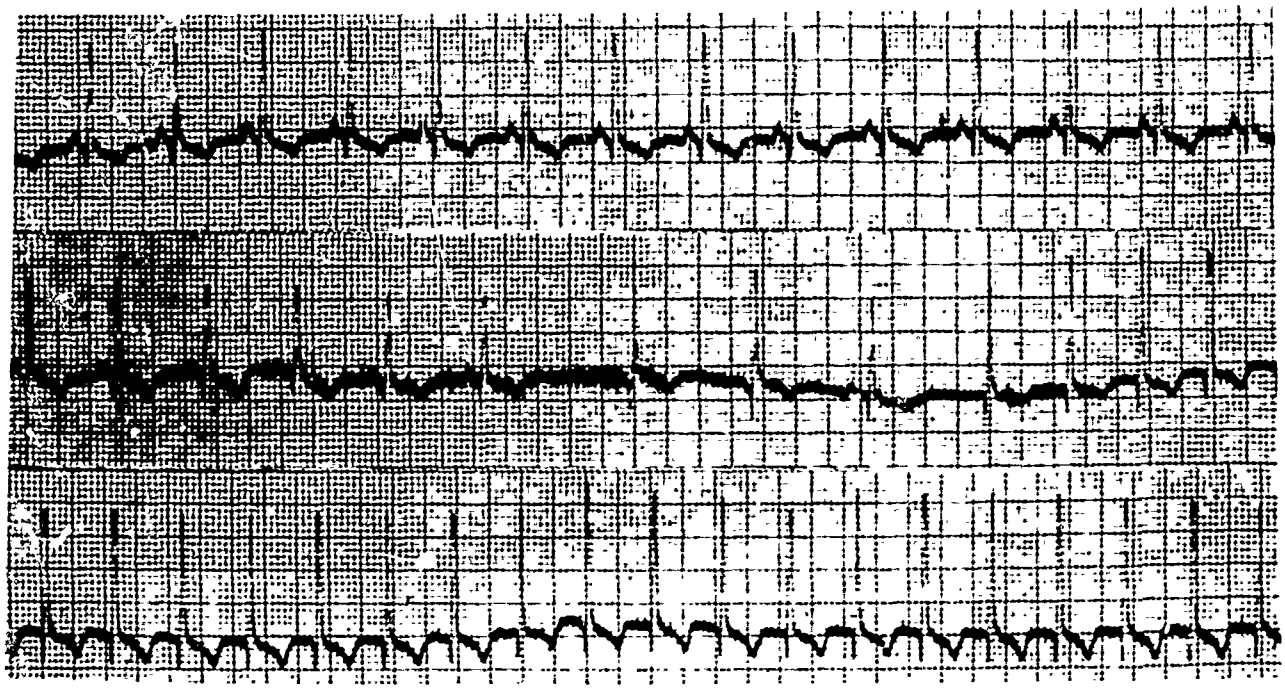


FIGURE 6

Subject 41: Lead II demonstrates nodal rhythm and burst of nodal tachycardia induced by breath-holding after hyperventilation.

as they obviously represent a somewhat younger age group. It is well known that differences in vagotonia occur with different age groups. Marked sinus arrhythmia is more prone to occur in the younger individual. Whether this is a significant difference in the two groups studied to date is not known.

It is apparent from the study of this small group of aviation cadets that the incidence of clinical syncope in cadet applicants is considerably higher than has been previously supposed. Much needs to be done before any statement can be made concerning selection technics to predict the future occurrence of syncope in any individual. It is highly significant that 14 individuals who presented experimental syncope by currently used procedures had apparently never experienced syncope on any other occasion. On the other hand, subjects who had previously experienced syncope could not be identified by use of these experimental procedures. An individual may be temporarily syncope-prone (e.g., as a result of recent excessive rapid weight loss) and not be syncope-prone subsequently.

CONCLUSION

Fifty young, apparently healthy aviation cadets in preflight training were evaluated with regard to syncope. Thirty percent of these subjects admitted that they had experienced previous, undisclosed syncope, when they were not in fear of detection or removal from the training program. Twenty-one of the cadets experienced experimental syncope during the syncope procedures. Cardiac arrhythmia was frequently associated with the syncopal episode and cardiac arrhythmia was frequently induced by respiratory maneuvers without syncope. Intravenous administration of atropine apparently prevented recurrence of cardiac arrhythmia induced by respiratory maneuvers. Significant cardiac arrhythmia was also noted in simple orthostatic syncope.

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